RESPONSES TO COMMENTS SUBMITTED BY THE CHEMICAL INDUSTRY COUNCIL OF CALIFORNIA (CICC).

I. OEHHA should provide a more transparent prioritization process.

Comment 1: The Draft Document does not clearly lay out OEHHA's prioritization process. Despite OEHHA's efforts to describe the prioritization procedures and steps in the Draft Document, it is difficult to understand how the final eleven chemicals were selected from the pool of over 200 TACs.

Response: OEHHA regrets that the commenter finds the description of the process hard to follow. The introductory section of the document lays out the basis for the prioritization, which is admittedly a complex process, in which a combination of arithmetical tools and scientific judgment were used on an unavoidably limited basis of evidence. We will attempt to clarify the process in our revision of the document. One point that seems to be confusing readers is that the initial prioritization based on exposure measures and readily available toxicity criteria does not incorporate specific information as to potential for differential impacts on infants and children. This is so because, aside from the developmental toxicity studies, most of the health criteria are based on endpoints seen in either occupational epidemiology studies or toxicological studies on mature animals. Thus, the initial prioritization had limited utility in directing OEHHA to the final proposal.

Comment 2: Most of the Draft Document is dedicated to a general review of the factors influencing why infants and children might be more susceptible than adults. Only about six pages of the 56page Draft Document, however, actually describe the prioritization process and its results.

Response: OEHHA considered it important to delineate the types of effects where differential impacts on children might reasonably be anticipated, since this consideration would figure largely in the final overall evaluation.

Comment 3: The Draft Document describes a six-step process, but this description does not provide enough detail to understand how OEHHA's selection process, going from 200 down to 5 chemicals.

For example, the Draft Document states that data for ranking based on existing health criteria and ambient air concentration data were available for only 90 of the more than 200 TACs. In other words, more than half of the TACs had insufficient data on which to base a ranking. Yet, it is unclear how many chemicals were excluded simply because they did not have air monitoring data, when later in the prioritization process, the decision was "heavily influenced by the toxicity of the compounds and less so by the estimated exposures to the compounds." Later, the Draft Document indicates that the entire list of TACs (not just those with air monitoring data) were reviewed to look for any chemical that should obviously be included based on existing knowledge of the toxicity of the TAC. This is confusing.

Response: Again, OEHHA regrets that the commenter has become confused by what is, admittedly, a complex process limited in some cases by inadequate data.

As explained on page 3, section II A paragraph 3, OEHHA used current approved chronic RELs when available and, in other cases, either used health protective levels from the earlier CAPCOA document or used draft RELs, which have been published but not approved. We then determined the ratios of these values to available concentrations in air. Thus, many TACs were excluded from consideration via this mechanism because the California Air Resources Board did not report ambient levels for these compounds. In the vast majority of these cases it appears that the reason for this data lack is because ambient levels in California are in fact negligible or zero. However, as noted on page 4

(section II A paragraph 5) OEHHA did examine the entire list of TACs to ensure that no important TACs were excluded from consideration merely due to inadequate ambient air data. In fact some materials (notably acrolein) for which measurement difficulties prevent accurate determination of overall ambient air levels, but for which there are reasonable grounds for expecting significant exposures, were included for further consideration, as were some (such as lead and vinyl chloride) where ambient air levels are low, but significant exposure "hot spots" are known to occur.

The law requires OEHHA to take into account public exposures to TACs in developing the list. Clearly, one method to do this is to evaluate available ambient concentration data. This is one of the factors we considered. However, due to the limits of the available ambient concentration data, we also considered whether public exposure was known to occur for those chemicals for which we could not identify measured ambient levels. We did this through consultation with the Air Resources Board and their emissions inventory databases.

Comment 4: It is also unclear how the rankings produced by step 2 (non-cancer toxicity) were combined with the rankings produced by step 3 (cancer). This appears to be an important step since it was used to narrow the number of candidate chemicals from 90 to 34.

Response: The approach used to combine cancer and non-cancer rankings is described in Section IIa, paragraph 4 (pages 3-4). This process partly used an arithmetical device, but also applied scientific judgment to arrive at a reasonable overall ranking. As noted in Section IIA, paragraph 5, this ranking was a contributor to the selection of 35 (not 34 ; that was an error in the text of the report) candidates, but was not the only input to that selection. As noted in response to comment 3, the overall information base on the toxicity of the TAC factored heavily into determining which of the 90 compounds should be followed more closely for this initial prioritization. It is also important to note that this is only the first listing. The list will be updated in coming years.

Comment 5: In contrast to the earlier steps, the criteria described in step 6 to narrow the number of candidates from 34 to the final candidate chemicals are clear. The first of these criteria was: "Any evidence indicating that infants and children may be more susceptible to the toxicological effects associated with that TAC than adults. The strength of this evidence was weighted heavily in this initial selection of eleven TACs that disproportionately impact children." (OEHHA. document, p. 5)

An important question arises as to whether other chemicals would have made it to the final eleven had these criteria been applied earlier in the process. Apparently these criteria were not applied in steps 1-4 since the Draft Document states these rankings were based on "existing health criteria and ambient air concentration data, but did not include information on differential sensitivity of infants and children." Since the purpose of prioritization is to develop "a list of up to five chemicals identified as Toxic Air Contaminants (TACs) that may cause infants and children to be especially susceptible to illness," we believe OEHHA should have applied these step 6 criteria earlier in the process.

Response: Since in most cases toxicological data specific to impacts on infants and children have not been collated for the TACs, and the available time and resources did not permit a full evaluation for all 200 TACS, OEHHA was obliged to take a sequential approach to the analysis. A further limitation was discovered in that even for the 35 agents selected for more detailed consideration based on both exposure and known toxicity of the chemical, appropriate data simply do not exist in several cases to adequately assess whether infants and children would be more impacted than adults. Our initial prioritization included evaluating information on exposure and the resulting estimated risks from existing ambient air concentrations to help focus on those TACs most likely to have an impact. However, as noted at several points in the OEHHA document, attempts were made to review the TAC list in its entirety, in consultation with ARB, to avoid any obvious omissions. Thus, the 6 criteria laid out were also used in principal in paring down the list from approximately 200 TACs to 35.

Comment 6: It is also unclear how OEHHA considered the various mechanisms of toxicity and their relative importance in the rankings. The clear intent of the Act is to lead to appropriate control measures for the identified TACs, which intuitively would be air emission sources. Therefore, it seems that appropriate consideration should be given to the compounds whose mechanisms of toxicity result from air exposure. For example, what would be the point of identifying a highly potent TAC that has food intake as it's major route of exposure, and the exposure via food intake is not related to ambient air concentrations of the TAC? There may be significant children's health concerns associated with the TAC but controlling ambient concentrations of the TAC will not affect the primary route of exposure. There is no clear statement in the Draft Document that air exposures were considered more important than other exposure mechanisms and that this premise was used in the prioritization process.

Response: OEHHA agrees on the importance of considering toxicological mechanisms in this prioritization process. Indeed, that was the reason for the extensive discussion of mechanisms for differential impacts on infants and children that the commenter noted under Comment 2. In addition, discussions at various points in the document, and particularly in Section IIIA (page 10 et seq.), make it clear that the primary exposure of interest is via air, as mandated by the Statute. That is why we looked at data on ambient concentrations in air as well as emissions from stationary and mobile sources. However, as the commenter points out, there are complexities in evaluating the contributions by different routes. These include (for example) the possibility of indirect exposure to initially airborne pollutants via food, and inhalation exposure to materials from contaminated soils or sediments via airborne dust. Since these considerations tend to be compound specific, they are addressed in the summaries for specific agents, when data were available. We noted under the 6 criteria, that exposures via noninhalation pathways were weighed in considering which chemicals to place on the list.

Comment 7: Although we believe OEHHA should more clearly explain its prioritization process, we feel it would be unnecessary and counter-productive to provide a detailed description of how each of the 200 chemicals are ranked. In fact, we strongly agree with OEHHA's decision to not provide a formal ranking of all 200 chemicals. But, it is important that the process be sufficiently transparent so that others, using the same methodology, develop a similar final list. Perhaps there would be value in showing in greater detail how the ranking process worked at each step for the 11 chemicals that were identified as either Tier 1 or 2.

Response: OEHHA acknowledges the commenter's agreement with a stepwise process, which was also delineated in the response to comment 5. OEHHA has attempted to explain the methodology used as far as possible, and to invite input from various sources, within the constraints imposed by the mandated timetable. Our intent is to identify TACs that may disproportionately impact infants and children. If chemical-specific information on the substances identified in Table 1 was missed that will help us identify potential for disproportionate impacts, we hope this comes to our attention during the public and peer review process. We have tried to provide information regarding the choice of the 11 chemicals in Table 1. In that table, we indicate where widespread exposure to the chemical was a concern in addition to the toxicological information, and which choices were driven primarily by the toxicological information.

Comment 8: In addition to more fully describing the prioritization process, we urge OEHHA, when it publishes the final list of up to five chemicals, to clearly communicate what the list is and what it is not. By identifying a chemical (or substance) as a priority TAC, OEHHA must clarify that it has not made a determination that any uses of the listed chemical pose significant risks to children's health. The level of potential risk to children will be determined as part of the implementation of the Children's Environmental Health Act. It is important for OEHHA to be very clear that for any given chemical identified as Tier 1, OEHHA may determine that reasonably anticipated

exposures and risks from expected uses do not pose any unique concerns for children's health and safety.

Response: OEHHA appreciates this concern; however, it must be pointed out that the current activity of establishing a list of TACs that may impact children is a hazard identification process, rather than a dose-response assessment. We also look forward to the findings of the Scientific Review Panel and their input into which chemicals should be on the list and any caveats they believe are necessary..

II. The brief descriptions of the eleven Tier 1 and 2 TACs in the Draft Document fail to provide sufficient explanation for why they were assigned the highest priority.

Comment 1: Although the Draft Document provides a brief description of the reason why each of the final eleven TACs were assigned to Tier 1 or 2 (OEHHA Document, pp 6-8), the explanations are insufficient. For example, the Draft Document states that benzene, the first chemical in Tier 1, was selected because "... it is leukemogenic in children and because of widespread exposure. Parental exposure to benzene is associated with elevated leukemia risk in the children indicating a heritable mutation occurs in germ cells. Thus, infants and children are susceptible subpopulations for this effect. Leukemia is the major form of childhood cancer." In other words, benzene was chosen because it causes leukemia and leukemia is the major form of childhood cancer.

There is no evidence, however, that children are more susceptible to benzene-induced leukemia than are adults. Recent scientific research, including the results of a recent study in Scotland, suggest that most childhood leukemia is due to an unidentified infectious agent, which might explain why we often see clusters of childhood leukemia. In cats, we know that leukemia is caused by the feline leukemia virus.

Response: As noted on page 8, greater detail for choosing the candidate chemicals for the list is provided in the toxicity summaries in Appendix B. As described in the draft benzene toxicity summary in Appendix B, there is a good deal of evidence to believe that

early life exposures to benzene may result in increased lifetime leukemia rates relative to adult exposures. With respect to the possible association of preconceptual parental exposure to benzene and elevated risk of childhood leukemia (implying a heritable mutation), the draft in all cases describes the evidence as suggestive. OEHHA appreciates the reference to additional studies not cited in the draft.

Comment 2: Besides arriving at an erroneous conclusion without substantiating data, what is the harm of elevating benzene to a high priority, even if it is not a major contributor to childhood leukemia? The harm is that we will focus our valuable resources on the wrong solution. Childhood leukemia is a very serious problem, and we cannot afford to waste limited resources going down the wrong path. The Draft Document leaves readers with the impression that (1) benzene is the main cause of childhood leukemia and (2) children are more susceptible to benzene-induced leukemia. Neither has been proven, and alternative hypotheses are at least equally plausible. But, the Draft Document does not mention any of this. The Draft Document should be more balanced in its description of benzene. Otherwise, people will be erroneously led to believe that we can dramatically reduce childhood leukemia simply by lowering ambient levels of benzene.

Response: The draft document is intended as technical analysis and background for the review to be made by the Scientific Review Panel for Toxic Air Contaminants, rather than a presentation in the popular media. OEHHA finds it implausible that the Panel members would suffer from misapprehensions of the type the commenter describes. In the interests of brevity, the authors focused on data having some bearing on the issues of differential sensitivity. However, they attempted to make clear that much of this evidence is preliminary and that not all the available evidence supports the presumption that benzene (or the other prioritized TACs) may differentially impact infants and children. Nowhere in the draft report do we state or imply that benzene is the major cause of childhood leukemia.

Comment 3: There are other examples where critical information was not included in the explanations for why certain chemicals were selected. For example, the Draft Document provided a lengthy review of microsomal P-450 enzyme formation in infants and children. The Draft Document correctly notes that many of these important enzymes are not fully developed in infants and children. Yet, it does not appear to take this information into account in its prioritization of chemicals. Many chemicals require these enzymes for metabolic activation. In other words, for certain chemicals (e.g., vinyl chloride), the parent molecule is not the toxic agent, and the molecule must be metabolically activated to form the toxic metabolite or intermediate. If the enzymes responsible for metabolic activation are not fully developed in infants and children, they should be less susceptible, not more susceptible, to chemicals that require metabolic activation. The Draft Document is quick to point out plausible mechanisms that would make infants and children more susceptible, but it fails to mention other plausible mechanisms that would render infants and children less susceptible to the very same chemicals.

Response: OEHHA has attempted to include the relevant evidence for the specific TACs selected in the toxicological summaries. In the particular case of vinyl chloride, the most striking feature of the data is the objective evidence (in animal studies by Drew et al., 1983 and Maltoni et al., 1981, cited in the OEHHA summary) that younger organisms are more susceptible to the carcinogenic effect of this chemical. OEHHA is therefore giving this specific evidence greater weight than any hypothesis based on general pharmacokinetic principles. However it should also be noted that the simplistic expectation that sensitivity to procarcinogens of the fetus or neonate should be lower because of lower cytochrome P450 levels has been clearly refuted in several cases where, although fetal or neonatal oxygenase activities are underdeveloped, the corresponding Phase II (detoxification) enzyme systems are even less active relative to the adult. A specific illustration of this may be found in the several reports of enzyme activities, toxicity and DNA adducts in fetal or neonatal rodents cited in the summary on benzo[a]pyrene and other PAHs.

III. OEHHA should assign priority levels on the basis of the "weight of the scientific evidence."

Comment 1: In general, OEHHA should use a weight of evidence process to evaluate the scientific data regarding the differential susceptibility of children. Determinations cannot be based on speculation. The prioritization and identification processes employed in the Draft Document will be equated with causality, yet the Draft Document does not follow a transparent and consistent procedure based on the well-established scientific principles of toxicology and epidemiology to examine the hypothesis of causality. Instead of systematically employing the Bradford-Hill criteria for causality for epidemiological data, the Draft Document has arbitrarily and inappropriately relied on speculation to make inferences. In several instances, the Draft Document appears to have inferred that a substance disproportionately impacts children because it is associated with a health effect in animal studies or in occupational studies of adults, when there is absolutely no scientific evidence regarding actual effects of the substance in children. The mixing of scientific findings and speculation implies a greater scientific certainty in the prioritization process than is actually supported by the evidence.

Response: OEHHA has been careful to apply the weight of evidence principle in evaluating the available scientific data on differential impacts of TAC on infants and children. Unfortunately this evidence is not as complete as one would like on many of the points at issue. OEHHA therefore has carefully considered all the available evidence, including mechanistic considerations and inferences from knowledge of general biological phenomena, where these could contribute to the final conclusion. OEHHA disagrees with the comment in its rejection of the use of extrapolation from animal models in the evaluation. Although use of animal data clearly involves certain uncertainties, to ignore it would dismiss almost all of the modern science of toxicology. The well-known Bradford-Hill criteria for identifying causality provide guidance as to the types of evidence that may be contributory or necessary (but not, in isolation, sufficient) in concluding that a causal association may exist: they also make it clear that such a conclusion may be more or less definite depending on the quality and extent of the evidence, and is unlikely to be absolute and irrefutable except in the most extreme and

well-studied cases. OEHHA rejects the comments' conclusion that the document used "speculation" to integrate all the available data (sparse as they are in some cases, either for or against the existence of a differential effect) into the overall evaluation of the weight of evidence. The law requires that we evaluate existing TACs for their potential to cause impacts on children, and does not require that we wait until impacts are clearly measured.

IV. OEHHA should amend the criteria for prioritization to give a higher priority to those chemicals known to have (rather than suspected to have) disproportionate impacts on infants and children.

Comment 1: Chemicals ***known*** to have disproportionate impacts on infants and children should be given a higher priority than chemicals merely ***suspected*** to have a disproportionate impact. For example, the increased susceptibility of infants and children to lead is well established, and lead is appropriately identified as a Tier 1 TAC in the Draft Document. OEHHA identified the major reason why lead was chosen as: "Children are the most susceptible subpopulation due to developmental neurotoxicity."

Response: OEHHA agrees that definite evidence of disproportionate impact on infants and children (or, in animal studies, on juvenile life stages) should be accorded greater weight than suspected impacts based on analogy or mechanistic interpretations. As noted, this was a major consideration for the prioritization of lead: OEHHA assures the commenter that this consideration was also applied in the other cases included in the process used to develop the proposed list.

Comment 2: Mercury is another example of a substance where the increased susceptibility of infants and children has been well documented. However, in contrast to lead, mercury appears in the Draft Document as a Tier 2 TAC. The major reason why mercury was chosen was exactly the same as it was for lead: "Children are the most susceptible subpopulation due to developmental neurotoxicity." The rationale for affording mercury a lower priority than lead is not apparent in the Draft Document.

Response: It is true that both mercury and lead were selected due to their disproportionate effects on children's health. However, in California the ambient levels of mercury are approximately ten-fold lower than lead (1.5 vs. 14 ng/m^3, respectively) and the primary route of exposure for mercury is via ingestion of mercury-contaminated fish. For these reasons OEHHA considered mercury less of an airborne health hazard and thus of lower priority than lead.

Comment 3: Mercury should be afforded a higher priority, and it should replace one of several Tier 1 TACs that are only suspected of having a disproportionate impact on infants and children. California has higher levels of mercury than many other states due to the historical presence of large mercury mines along its coastal mountain range. At one point, the largest mercury mine in the world was located in the San Francisco Bay Area. To this day, mercury mine tailings continue to contaminate the local air and streams, which flow directly into the San Francisco Bay. Since SB 25 is a California law, it makes sense to focus on the issues of greatest relevance to California.

Response: OEHHA agrees that mercury contamination from mining operations is an important concern, but in California it is largely limited to pollution of water. Mercury in the air occurs mainly from the burning of fossil fuels, notably coal, whose use is much less prevalent in California than elsewhere in the country. The United States Geological Survey (USGS) monitors mining sites and other potential natural sources of mercury such as geysers. The contribution of these sites to airborne mercury is very limited. Thus while OEHHA considers mercury an important neurodevelopmental toxicant, as an airborne toxicant it is assigned to Tier 2 due to its low ambient levels, and the minor role of air as a transport medium for mercury in the specific conditions present in California.

Comment 4: The Draft Document should be revised to clearly differentiate between those chemicals that are known to have a disproportionate impact on infants and children compared to those suspected to have such an effect. There are a limited number of

chemicals that have been clearly shown to impact infants and children disproportionately. It is important to distinguish between chemicals we are sure have a disproportionate effect vs. chemicals where we are suspicious (but not sure). The Draft Document must differentiate between fact and hypothesis, so decisions can be based on the best available information.

The authors of the Draft Document indicate that chemical-specific references on children related susceptibility are not easy to find. Some of the final selections were based on "plausible mechanisms" rather than "known information." This is another reason why it is especially important to clearly communicate what we know and what we don't know.

Finally, in several places, the Draft Document uses imprecise and confusing terminology. In some places, it describes the final list of 5 chemicals as chemicals that "may" or "potentially" cause infants and children to be especially susceptible to illness. In other places, the Draft Document describes the list as "the list of TACs that differentially impact infants and children" and "five TACs that disproportionately impact children." (OEHHA document, page 4.) Chemicals that are known to disproportionately impact children should be described as such. But chemicals that are only suspected to disproportionately impact children should be described in less certain terms.

Response: OEHHA has attempted to make the level of confidence associated with its evaluations plain to the reader, and believes that this was achieved in the public review draft. However, the document will be reviewed and possibly revised in the light of these and other public comments and the findings of the Scientific Review Panel. If OEHHA finds any confusing or inaccurate descriptions of the level of confidence achieved in an evaluation, they will be corrected in the final draft. The commenter is referred to the text of the legislation (included as Appendix A of the public review draft of OEHHA's document) for a characterization of the precise status of the list. The law states that we are to develop a list of TACs "that may cause infants and children to be especially

susceptible to illness". Thus the list may contain some compounds for which the data are less clear than others. The law does not exclude any TAC from consideration.

Finally, the statute requires OEHHA to list "up to five" TACs that "may cause infants and children to be especially susceptible to illness". The words "may cause" are key in this sentence; the legislature recognized that we may not have all the data we would like to show beyond doubt that specified substances are more toxic in infants and children than in adults. Thus suggestive evidence may be used to list a substance under this statute. OEHHA has attempted to make it clear to the reader where we thought the data were "suggestive" as opposed to where the data are clear (as in the case of lead). Certainty unfortunately only exists in those cases where substantial harm to children has already occurred (e.g., lead and mercury). The law's primary intent is to ensure the safety of infants and children. We have followed the criteria specified in the law to identify those TACs that may pose risks to infants and children especially.

V. OEHHA should assign a higher priority to chemicals for which air is the major source of exposure and a lower priority to chemicals for which air is a relatively insignificant source of exposure.

Comment 1: The Children's Environmental Health Protection Act focuses on infant's and children's TAC exposures through the air. Indeed the Act requires this prioritization to ensure that the state's air quality standards and airborne toxic control measures adequately protect the health of infants and children. Section I of the Act clearly highlights the intent to address air and, in particular, inhalation exposures by citing specific respiratory diseases and the susceptibility of infants and children to mechanisms of TAC's due to differences in respiratory function. Although this ***seems*** clear in the Act, the Draft Document does not indicate that this consideration was important when compiling data on the toxicity of TAC's. This consideration is critical in that the Tier I TAC's should be those that can be affected through air quality standards and airborne toxic control measures. Those TAC's with major exposure mechanisms via other media will need to be controlled though other programs.

The Draft Document should give a higher priority to chemicals for which air is the primary source of exposure. Conversely, chemicals for which air is an insignificant relative source of exposure should be afforded a lower priority.

Response: OEHHA agrees that the intent of the legislation was to address airborne pollutants specifically, and higher priority was therefore given to chemicals for which air is either a source of direct exposure resulting in significant potential risk, or an important link in the transport mechanisms leading to human exposure via other media. The consideration of ambient exposure is discussed on pages 3-8 and in Appendix B for each specific chemical.This consideration is exemplified in our response to the comments about mercury and dioxin. Since the intent of the process is to address risk from toxic air contaminants, it is not strictly relevant whether a substantial risk also exists via media or routes completely unrelated to airborne pollution, or whether this risk is larger or smaller than that which is related to air as a transport or exposure medium. However, in practice all these issues are likely to be related and were included in the overall consideration.

Comment 2: For example, PAHs were designated as Tier 1 TACs due to developmental effects, genotoxicity and lung cancer. If the air is a major source of exposure to PAHs, then it would be appropriate to assign PAHs a high priority. However, there are many common sources of exposure to PAHs, including certain FDA-approved anti-dandruff shampoos, as well as a variety of charcoal-broiled foods. If these other sources dwarf the amount of exposure to PAHs from air pollution, then PAHs should be given a lower priority.

Response: PAHs are considered important air contaminants and for this reason have been evaluated and identified as Toxic Air Contaminants. There is substantial evidence, documented in OEHHA's toxicity summary for benzo[*a*]pyrene and other PAHs, that airborne exposures to PAHs may result in substantial additional risk to public health, especially in urban environments and in local situations where substantial PAH exposures occur. The ARB has developed test methods to measure these contaminants on a regular

basis. The risk from airborne PAH exposures are considered very important. Risks of PAHs in foods have been separately studied and reported. It is believed that certain specific population groups with diets unlike those typical in California do in fact show enhanced risks (such as that for stomach cancer), but the answer to whether such risks are substantial for California residents is unknown and controversial. The continued approval of the anti-dandruff shampoos by US FDA (which has been the subject of some discussion recently) implies that that agency at least considers that the associated risks, if any, of that application are outweighed by its benefits.

Comment 3: In the case of dioxin, the major route of exposure is dietary ingestion, not inhalation. Over 90% of a person's daily exposure to dioxin occurs from dietary intake, primarily in meat, fish and dairy products. Exposure to dioxin by inhalation of ambient air is insignificant, but airborne dioxin may lead to contamination of sources of food. It would make sense to assign dioxin a TAC priority level based on the overall importance of airborne dioxin on exposure levels in food.

Response: OEHHA agrees with the comment that exposure to dioxins is largely from dietary intake. However, newly formed dioxins from industrial or other anthropogenic sources, and from natural sources such as forest fires, are often released into the atmosphere in the first instance and subsequently deposit to the surface and enter the food chain. Dioxins and related persistent chlorinated pollutants are present in all media including sediment, soil, and biota, and are likely to be recirculated into the atmosphere. Because of their persistent and bioaccumulative properties, dioxins originally released into the air are found in all levels of the food chain, including humans. There is increasing evidence that background levels of dioxin cause deleterious neurobehavioral, reproductive, and immunological effects in newborns and infants exposed *in utero* or via lactation. These effects are reported to last until and in some case beyond school age. Therefore airborne dioxin is an environmental contaminant that requires close monitoring. OEHHA has assigned dioxin a TAC priority level based on the overall importance of airborne dioxin, including consideration of exposure levels in food.

SMOKING INITIATION AND OLD GOLD CIGARETTES

DISCOVERY DEPOSITION

Q: Can you tell me why you were smoking prior to 1950?

A: Well, it's probably because the older guys smoked.

Q: So you saw – go ahead, finish your answer.

A: And maybe when I used to go to the movies a lot, I would see them in the movies.

Q: You would see the older guys smoking or people in the movies?

A: The movie stars.

Q: So you indicated that you had seen older kids smoking cigarettes prior to 1950, is that right?

A: Oh, yes ...

Q: Other than seeing the older kids smoke and wanting to be like the older kids and seeing cigarettes being smoked in the movies, is there anything else that you feel led you to smoke cigarettes prior to 1950?

A: *No, sir.*

(Exhibit A, David Tompkin 11/8/94 Deposition Transcript at 42-43) (emphasis added).

Q: Do you feel that's one of the reasons you may have smoked Old Golds because it was a brand that your brother may have smoked?

A: Yes.

Q: Can you think of any other reason that led you to choose Old Gold as a brand back in 1950 to 1955?

A: No.

(Exhibit A, David Tompkin 11/8/94 Deposition Transcript at 48).

Q: Now I think you told us that your brother Gilbert used to smoked Old Gold cigarettes?

A: Yes, sir.

Q: Okay. And that's the reason you smoked Old Gold cigarettes, isn't that right?

A: Yes.

Q: Okay. And there is no other reason that you smoked Gold – Old Gold cigarettes, isn't that true?

A: Not that I recall now.

Q: Okay. And you don't know why you smoked Philip Morris cigarettes, isn't that true?

A: Not really.

(Exhibit B, David Tompkin 11/18/94 Deposition Transcript at 122).